AMENDMENTS TO THE CLAIMS:

Claims 1 through 26 (canceled)

27. (original) A method comprising evaluating a G protein coupled receptor agonism or antagonism of a compound by a bead-based flow cytometric process comprising contacting the compound with beads conjugated to a G protein coupled receptor ligand which would result in a detectable G protein coupled receptor ligand-receptor complex, to determine the existence of an interaction or an an absence of an interaction with said receptor, wherein the extent to which the compound competetes with said ligand-receptor complex determines that a compound is an agonist or an antagonist of said G protein coupled receptor.

28. (original) A method comprising evaluating a β2-adrenergic receptor agonism or antagonism of a compound by a bead-based flow cytometric process comprising contacting the compound with beads conjugated to a β2-adrenergic receptor ligand which would result in a detectable β2-adrenergic receptor ligand-receptor complex, to determine the existence of an interaction or an an absence of an interaction with said receptor, wherein the extent to which the compound competes with said ligand-receptor complex determines that a compound is an agonist or an antagonist of said β2-adrenergic receptor.

29. (original) A method of claim 28, wherein the beads are dihydroalprenolol-conjugated beads.

30. (original) A method comprising evaluating a G-protein receptor agonism or partial agonism of a compound in a bead based high throughput screening system comprising a) contacting the compound and solubilized detectable G protein coupled receptor with G protein beads, each of said G-protein beads comprising epitope-recognizing beads having an epitope-tagged heterotrimeric G protein bound thereto; and b) determining whether a ternary complex between said G protein coupled receptor and said G protein occurs, wherein an interaction between receptor and G protein evidences that said compound is an agonist or partial agonist of said G protein coupled receptor.

31. (original) A method of claim 30, wherein the G protein receptor is the β2-adrenergic receptor, said receptor contains a fluorescent moiety, and the interaction between said receptor and said G protein evidences that said compound is an agonist of said receptor.

32. (original) A method of claim 30, wherein said the detectable moiety is a any fluorescent protein.

33. (original) A method of claim 31, wherein the β2-adrenergic receptor is a β2AR-GFP fusion protein.

34. (original) A method of claim 30, wherein detectable ternary complex levels are used to generate dose-response curves that are indicative of the compound's β2-adrenergic receptor agonism or antagonism.

35. (original) A method of claim 30, wherein GTPγS-induced activation rates for the detectable ternary complex are determined and wherein compounds that are β2-adrenergic receptor agonists or partial β2-adrenergic receptor agonists have approximately equal GTPγS-induced activation rates.

36. (original) A method comprising evaluating the relative G protein receptor agonism or partial agonism a compound by a flow cytometric process comprising contacting the compound and soluble detectable G protein receptor with beads conjugated to epitope-recognizing beads having a heterotrimeric G protein bound thereto, wherein an agonist or partial agonist compound binds to G protein receptor to form a compound-receptor complex and said compound-receptor complex binds to said bound G protein to form a detectable ternary complex indicative of the compound's G protein receptor agonism or antagonism.

37. (original) A method of claim 36, wherein the G protein receptor is a β2-adrenergic receptor containing a fluorescent moiety.

38. (original) A method of claim 37, wherein the fluorescent moiety is a GFP or a RFP fused to said G protein receptor.

39. (original) A method of claim 36, wherein the detectable β2-adrenergic receptor is a β2AR-GFP fusion protein.

40. (original) A method of claim 36, wherein GTPγS-induced activation rates for the detectable ternary complex are determined and wherein compounds that are β2-adrenergic receptor agonists or partial β2-adrenergic receptor agonists have approximately equal GTPγS-induced activation rates.

41. (original) A method of evaluating a library of compounds comprising:
selecting a plurality of compounds from the library;
evaluating the relative β2-adrenergic receptor agonism of each selected compound by a flow cytometric process comprising contacting the compound with beads conjugated to a β2-adrenergic receptor-detectable moiety complex, wherein the extent to which the compound complexes with the β2-adrenergic receptor-detectable moiety complex to form a detectable ternary complex is determined by measuring detectable ternary complex levels and detectable ternary complex levels are indicative of the compound's β2-adrenergic receptor agonism or antagonism; and
evaluating a differentiation state or a metabolic parameter of the cell or organism.

42. (original) A method comprising evaluating the relative G protein receptor agonism, antagonism or inactivity of a compound for a G protein coupled receptor (GPCR) in a single sample by a flow cytometric process comprising the steps of (a) providing a sample suspension containing a detectable GPCR, a set of G protein beads which will form a ternary complex with said detectable GPCR in the presence of an agonist or partial agonist, and a set of ligand beads which will bind to said detectable GPCR, said set of G-protein beads comprising epitope-recognizing beads having a heterotrimeric G protein bound thereto; (b) mixing said sample suspension with said compound; and (c) detecting the formation or absence of formation of a complex between said compound and said detectable GPCR, wherein a GPCR antagonist prevents binding of said detectable GPCR to said G protein beads by preventing ternary complex formation and prevents binding of said detectable GPCR to said ligand bead; a GPCR agonist allows binding of said detectable GPCR to said G protein beads by forming a ternary complex but prevents binding of said detectable GPCR to said ligand bead; and an inactive compound prevents binding of said detectable GPCR to said

G protein beads by not promoting ternary complex formation but allows binding of said detectable GPCR to said ligand bead.

43. (original) A method of claim 42, wherein the G protein coupled receptor is a β2-adrenergic receptor containing a fluorescent moiety.

44. (original) A method of claim 42, wherein the fluorescent moiety is any fluorescent protein fused to said G protein coupled receptor.

45. (original) A method of claim 43, wherein the detectable β2-adrenergic receptor is a β2AR-GFP fusion protein.

46. (original) The method of claim 42 wherein said G protein beads are modified with a fluorescent moiety.

47. (original) The method of claim 46 wherein said fluorescent moiety is Texas Red.

48. (original) A method comprising identifying agents useful in the treatment of a disease associated with G protein coupled receptor (GPCR) agonism or antagonism by determining an agent's GPCR agonism or antagonism by a flow cytometric process comprising: (a) providing a sample suspension containing a detectable GPCR, a set of G protein beads which will form a ternary complex with said detectable GPCR in the presence of an agonist or partial agonist, and a set of ligand beads which will bind to said detectable GPCR, said set of G-protein beads comprising epitope-recognizing beads having a heterotrimeric G protein bound thereto; (b) mixing said sample suspension with said agent; and (c) detecting the formation or absence of formation of a complex between said agent and said detectable GPCR, wherein a GPCR antagonist prevents binding of said detectable GPCR to said G protein beads by not promoting ternary complex formation and prevents binding of said GPCR to said ligand bead; and a GPCR agonist allows binding of said detectable GPCR to said G protein beads by forming a ternary complex but prevents binding of said detectable GPCR to said ligand bead.

49. (original) A method of claim 48, wherein the G protein receptor is a β2-adrenergic

receptor containing a fluorescent moiety.

50. (original) A method of claim 48, wherein the fluorescent moiety is any fluorescent protein fused to said G protein receptor.

51. (original) A method of claim 49, wherein the detectable β2-adrenergic receptor is a β2AR-GFP fusion protein.

52. (original) The method of claim 48 wherein said G protein beads are modified with a fluorescent moiety.

53. (original) The method of claim 46 wherein said fluorescent moiety is Texas Red.

54. (original) A method comprising evaluating a G protein coupled receptor agonism or antagonism of a compound by a bead-based flow cytometric process comprising contacting the compound with beads conjugated to a ligand which would result in a detectable G protein coupled receptor ligand-receptor complex, to determine the existence of an interaction or an an absence of an interaction with said detectable receptor, and comparing said interaction with said ligand-receptor complex with a ligand-receptor complex utilizing a known agonist or antagonist to determine that a compound is an agonist or an antagonist of said G protein coupled receptor.

55. (original) A method of evaluating a library of compounds comprising:
selecting a plurality of compounds from the library;
evaluating the relative β2-adrenergic receptor agonism or antagonism of each selected compound by a flow cytometric process comprising contacting the compound with beads conjugated to a ligand for a β2-adrenergic receptor-detectable moiety complex, wherein the extent to which the compound complexes with the β2-adrenergic receptor-detectable moiety and binds to said ligand conjugated beads is indicative of agonist or antagonist activity; and
evaluating a differentiation state or a metabolic parameter of the cell or organism.